



Clinical and Hematobiochemical Changes in Foals and Adult Horses with Upper and Lower Respiratory Tract Affections



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Abstract

EQUINE respiratory diseases represent a major health challenge for both young foals and adult horses. Therefore, this study aimed to evaluate and compare the respiratory affections in foal and adult horses with upper and lower respiratory diseases. A total number of 150 horses (30 control and 120 diseased) were subjected to clinical, hematological and biochemical examinations. The affected foals and horses were categorized according to clinical and ultrasonographic examination into upper respiratory tract affections (URT) and upper complicated with lower (UCL) respiratory affections. Blood and serum samples were collected from diseased and control foals and adult horses. The results showed significant decrease ($P < 0.05$) in RBCs, HCT, PLT, albumin, A/G, SOD and CAT activity with significant increase ($P < 0.05$) in WBCs, neutrophil, SAA, Hp, CRP, IL-6, TP, globulin, MDA in URT subgroups. While the UCL subgroups showed significant decrease in RBCs, Hb, HCT, PLT, albumin, A/G, SOD and CAT activity with significant increase ($P < 0.05$) in WBCs, neutrophil, lymphocytes, SAA, Hp, CRP, IL-6, TP, globulin and MDA. Moreover, there was significant change in WBCs, neutrophil, lymphocytes, HCT, PLT, SAA, Hp, CRP, IL-6, TP, Albumin, Globulin, A/G, SOD, MDA, and CAT between foals and adult horses. Thus, we concluded that timely intervention is crucial to prevent the progression of upper respiratory affections to lower respiratory complications, which can have more severe consequences on the horse's health. Moreover, early management of respiratory tract diseases in horses is recommended through regular monitoring and veterinary care.

Keywords: Foal, Horse, MDA, Respiratory, SAA.

Introduction

Equine respiratory diseases (ERDs) are considered the most common serious health problems affecting both young and adult horses. ERDs had an adverse effect on physiological performance and cause high morbidity and mortality rates [1]. They could be mentioned as the most important cause of illness and death in foals up to 12 months of age [1, 2]. Different factors have been involved to the occurrence of upper respiratory tract (URT) diseases, including young age, husbandry and management practices, inadequate biosecurity measures, environmental circumstances and immune-suppression due to exercise and transportation [3].

Adult horses frequently develop lower respiratory tract (LRT) infections, which can vary in severity from minor viral infections to serious complicated bacterial infections [4].

Lower airway bacterial colonisation can result from an initial viral URT infection that compromises mucociliary clearance mechanisms and suppresses local immunity within the lung transplant region. Consequently, opportunistic organisms colonisation from the upper airways is usually the source of lower airway infections in horses [5].

In contrast to neonates, whose bacterial pneumonia is frequently of haematogenous origin; adult horses get bacterial pneumonia mostly from inhaled or aspirated microorganisms from URT [6].

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As a result, the majority of adult respiratory infections begin on the respiratory mucosa's surface and eventually spread to the pulmonary parenchyma [7]. Consequently, in many episodes of pneumonia in horses, mixed aerobic and anaerobic bacteria are present, and a variety of opportunistic pathogens have been identified [6, 7].

Clinical signs commonly observed in equine respiratory diseases include cough, discharge from the nose or eyes, intolerance to exercise, respiratory distress, sneezing, abnormal respiratory noise, elevated breathing rate, abnormal respiration pattern, and systemic signs like depression, fever, and anorexia, especially in cases of infectious diseases [8]. Horse diseases have classically been diagnosed, tracked and prognosed using a range of inflammatory indicators in conjunction with alterations in several haematological and biochemical parameters [9].

The systemic and dynamic process known as acute phase response (APR) involves a variety of pathophysiological reactions including fever, leukocytosis, changes in hormones levels and depletion of muscle proteins. These responses work together to reduce tissue damage and speed up the healing process [10]. APR is triggered by certain plasma proteins called acute-phase proteins (APPs), which are mostly released by the liver under effect of proinflammatory cytokines specially interleukin-6 (IL-6) and offer early all-encompassing defence against shocks. This results in increased plasma concentrations of some known as (positive APPs) including serum amyloid A (SAA), Haptoglobin (Hp) and C-reactive protein (CRP). On the other hand, some proteins showed decrease in their levels known as (Negative APPs) including Albumin, which is the most abundant constitutive plasma protein [10,11].

Oxidative stress (OS) can be pathogenic, facilitating disease processes and stress responses or physiological, maintaining biological systems. Changes in OS biomarkers may indicate the extent of damage or the degree of deviation from normal. The interpretation of each oxidative stress marker will be more reliable in the diagnosis and helpful for comprehending the overall process if there is a complete shift in oxidative or anti-oxidative markers through a trustworthy indicator of alteration [12-14].

Furthermore, the respiratory tract is thought to be a primary source of oxidative injury. Evidence suggests that respiratory OS may contribute to the development of a number of respiratory disorders in horses, including exercise-induced pulmonary haemorrhage and recurrent airway obstruction. Also, it is believed to be connected to a few significant infectious illnesses in agricultural animals, like pneumonia and enteritis [15]. It has been demonstrated that in horses, the presence of systemic OS biomarkers correlates with cellular markers of airway inflammation [13]. Superoxide dismutase (SOD) is the first line of defence against reactive

oxygen species (ROS) and is essential in reducing OS [16]. One of the secondary products of lipid peroxidation is malondialdehyde (MDA), which is thought to be a marker for inflammation as well as lipid peroxidation associated with oxidative stress. It may be formed as a side reaction of prostaglandin production. Furthermore, illness may upset the balance of antioxidants and oxidants, which would lower Catalase (CAT) activity. Numerous studies showed a decline in SOD and CAT activity and elevated MDA in equine respiratory disorders [17, 18].

Therefore, the aim of the current work was to assess the clinical and hematobiochemical changes in upper and upper complicated with lower respiratory tract affections in young and adult horses. In addition, we hypothesized that acute phase proteins, pro-inflammatory cytokines and oxidant-antioxidant status could be different in both groups which is crucial for understanding the pathophysiology of these conditions and developing methods of diagnosis and treatment.

Material and Methods

Animals

The study was conducted on 150 horses aged (foals from 3 months to 1 year and adult from 2 to 6 years) with average body weight (85 kg for foals and 400 kg for adult horses) from private farms at Sharkia governorate and Giza governorate in the period from September 2022 until March 2023. Selected cases were examined physically and clinically for determination of body temperature, heart and respiratory rates as well as examination of mucous membranes, lymph nodes and auscultation of lung and heart sounds were conducted [19].

Selected animals were classified into two groups according to the age, the foal group and the adult horse group. Each group was subdivided into three subgroups; a control healthy subgroup, horses affected with upper respiratory affections only (URT subgroup) and horses that are affected with Upper respiratory affection complicated with lower respiratory affection (UCL subgroup). The occurrence of lower respiratory affections including pneumonia and consolidation was confirmed by the clinical examination of lung and chest ultrasonography. The animal experimental design is summarized in Table (1).

Ethical approval

The study was carried out with the Benha University Ethics Committee's approval with approval number (BUFVTM 36-09-23).

Blood and serum samples

Five ml of whole blood was withdrawn from jugular vein in a sterile vacuum tube (with EDTA) for hematological evaluation. An additional 5ml blood sample was drawn into a clean, dry test tube

without anticoagulant. The sample was then allowed to clot at room temperature and then centrifuged for ten minutes at 3000 rpm to extract clean, non-hemolyzed serum. The serum was then transferred into a clean, dry and sterilized Eppendorf tube and stored at -20 °C until it was needed for biochemical analysis.

Hematological analysis

Hematological analyzer (Model No. 93-91098-00-GF) was used to measure total leukocyte count (WBCs), total erythrocytic count (RBCs), Hb, HCT, PLTs count, and other hematological parameters [20].

Biochemical analysis

Serum proteins analysis

Spectrophotometric analysis was performed on serum total proteins (TP), albumin, and globulin utilizing test kits from Spectrum-diagnostic, Egypt. also, the acquired serum samples were used for the spectrophotometric detection of SAA, Hp, CRP and Interleukin- 6 (IL-6) by using commercial kits (Egyptian Company for Biotechnology (S.A.E). Obour city industrial area. Cairo. Egypt).

Oxidant –Antioxidant status analysis

Using commercially accessible test kits (Bio diagnostics, Cairo, Egypt), standard procedures were followed to detect SOD, MDA, CAT, and Total Antioxidant Capacity (TAC).

Statistical analysis

The statistical analysis was carried out using two-way ANOVA using SPSS, ver. 25 (IBM Corp. Released 2013). Multiple comparisons were carried out applying Duncan test. The significance level was set at $P < 0.05$.

Results

Clinical findings

In this study, clinical symptoms such as fever, elevated respiratory rate, cough, tachycardia, and thoracic pain were commonly reported in the UCL subgroup compared to the URT subgroup in both foals and adult horses. On the other hand, nasal discharge and submandibular lymphadenopathy were more frequent in the URT subgroup of foals and in the UCL subgroup of adult horses. Thoracic pain and ultrasonographic changes were predominantly observed in the UCL subgroup in both foals and adult horses (Table 2).

Foals and adult horses with URT exhibited signs of rhinitis, pharyngitis, laryngitis, and tracheitis. Some diseased animals in both foals and adult horses also showed guttural pouch empyema, characterized by purulent discharge from the guttural pouches (Fig. 1).

Clinical examination of the diseased animals revealed lethargy, inappetence, high body temperature in some cases, dry or moist cough, and various types of nasal discharge (Fig. 2). Additionally, some cases displayed upper respiratory signs along with lower respiratory issues such as pneumonia and bronchopneumonia, which were accompanied by grunting, thoracic pain, crackles, and wheezes. Auscultation and ultrasonography of certain diseased horses showed lung consolidation (Figs. 3,4 and 5) indicated by audible cardiac sounds in the lung field and the absence of normal vesicular lung sounds. This was in contrast to the findings in healthy control horses (Fig. 6).

Hematological findings

In the URT subgroups, there was significant ($P < 0.05$) reduction in RBCs, HCT and PLT in both foal and adult with significant ($P < 0.05$) increase in Neutrophil in foal and WBCs & Neutrophil in adult compared to the control group. While in the UCL subgroups, there was significant ($P < 0.05$) decrease in RBCs, Hb, HCT and PLT in foal and RBCs, Hb and PLT in adult compared to the control group. Additionally, there was significant ($P < 0.05$) increase in WBCs and Neutrophil in foal and WBCs, Neutrophil and lymphocytes in adult group compared to control groups.

However, there was significant ($P < 0.05$) decrease in Hb and significant ($P < 0.05$) increase in WBCs and neutrophil in the UCL subgroup of foal compared with URT subgroup in foal. As well as there was significant ($P < 0.05$) decrease in Hb with significant ($P < 0.05$) increase in WBCs, Neutrophil and lymphocytes in the UCL subgroup compared to URT subgroup in adults. Likewise, there was significant ($P < 0.05$) change in HCT, PLT, WBCS, Neutrophil and lymphocytes between foal and adult horses (Table 3).

Biochemical findings

There was significant ($P < 0.05$) elevation in SAA, Hp, CRP and IL-6 in URT and UCL subgroups in both foals and adult horses compared to control group. However, there was significant ($P < 0.05$) elevation in SAA, Hp, CRP and IL-6 in the UCL subgroups of both foal and adult compared to URT subgroups. Moreover, there was significant ($P < 0.05$) change in SAA, Hp, CRP and IL-6 between foals and adult horses (Table 4).

In the URT subgroups, there was significant ($P < 0.05$) decrease in albumin & A/G in both foal and adult groups with significant ($P < 0.05$) increase in globulin in foal and TP and globulin in adult group compared to control group. On the other hand, there was significant ($P < 0.05$) decrease in albumin and A/G and significant ($P < 0.05$) increase in globulin in UCL subgroup of foal with significant ($P < 0.05$) decrease in Albumin and A/G and significant

($P < 0.05$) increase in Globulin and TP in adult group compared to control. In addition, there was significant ($P < 0.05$) decrease in Albumin & A/G with significant ($P < 0.05$) increase in TP and Globulin in UCL subgroups of both foals and adults than URT subgroups. Moreover, there was significant ($P < 0.05$) change in TP, Albumin, Globulin and A/G between foal and adult horses (Table 5).

In the URT subgroups, there was significant ($P < 0.05$) decrease in SOD and CAT in both foal and adult groups with significant ($P < 0.05$) increase in MDA in both foal and adult group compared to the control group. While in the UCL subgroups, there was significant ($P < 0.05$) decrease in SOD, CAT in foals and adults with significant ($P < 0.05$) increase in MDA in both foals and adults compared to control subgroup. Moreover, there was significant ($P < 0.05$) decrease in SOD, CAT with significant ($P < 0.05$) increase in MDA in the UCL subgroup than URT subgroups. On the other hand, there was significant ($P < 0.05$) change in SOD, CAT and MDA between foal and adult horses groups (Table 6).

Discussion

Respiratory diseases are considered a major cause of time off work and poor performance for equines [21, 22]. A thorough history as well as a thorough physical and clinical examination are essential to the diagnostic assessment of ERDs [2]. To ascertain the degree and type of lower respiratory tract involvement, physical examination is essential. For the most part, this should comprise a rebreathing test, which is one of the most crucial diagnostic methods for evaluating pulmonary disease since it enables the identification of lower respiratory inflammation during auscultation and determines the existence and degree of lung involvement [7, 8, 23].

The changes detected in our study in most measured parameters were more prominent in adult group than foal group. This might be related to the foal's less developed immune system than of the adult's. Previous reports [24, 25] may strengthen this result. Also, Reuss and Cohen, [24] demonstrated that the pulmonary immune system in equine neonates is not mature at birth. Therefore, the susceptibility in foals and weanlings to pneumonia may be linked to decrease transfer of maternal antibodies and delay in the production of the foal's own antibodies. Therefore, they are susceptible to pathogens such as *Rhodococcus equi* infection while older foals are less susceptible and adult horses are resistant to infection with this bacterium unless they are immunocompromised. These results suggest that as foals get older, their respiratory tract's immune system develops. Furthermore, compared to peripheral blood, the development of the lung immune system appears to happen a little more slowly. Age-related differences in the immune

responses between adult horses and foals may also be related to antibody specificity and Th1 (cell-mediated) versus Th2 (humoral) immune responses. Therefore, given that the innate and adaptive immune responses of foals differ significantly from those of adult horses in terms of both content and magnitude, it is clear that we do not fully understand the mechanisms driving the development of the foals' immune systems. Under some circumstances, it has been reported that certain facts of the immunological response in a foal are similar to those in an adult horse. The foal immune response develops more quickly in some organs than in others. Furthermore, protective immunity against infection in horses lacks a functional description at the cellular, molecular, or physiological levels [27].

Additionally, our results showed that the changes in measured parameters were more obvious in the UCL subgroups than URT subgroups in both foal and adult and this was coincided with Couetil and Hawkins [28] and Fels *et al.* [29] who stated that severe and widespread lung illnesses affecting the distal airways are necessary to produce flow limitation. With increasing pneumonia severity, auscultation observations in the lung and trachea were more obvious and the volume of pleural effusion, the degree of pulmonary necrosis and the extent of lung involvement all correlate with the severity of clinical symptoms. In the present study, the clinical findings in most commonly described cases of the URT subgroup were in the line with previous reports [30, 31]. While the clinical finding in the UCL groups were agreed with previous reports [17, 23]. Large airway inflammation is demonstrated by the development of a cough during a rebreathing test. The abnormal breathing sounds suggest presence of lower respiratory inflammation, wheezes occurred as a result of small airway restriction while crackles occurred subsequent to the presence of fluid in the small airways. Furthermore, consolidated lung tissue and pleural effusion may be present in the presence of extremely loud airway noises or the lack of such sounds, and this was previous reports [7, 23].

A thorough assessment of the complete blood count (CBC) can provide insight into illness processes, their severity, and even diagnosis [20]. According to the haematological measures in the present study, anemia was indicated which was manifested with a decrease in Hb concentrations, HCT, and erythrocytic counts. This may be explained by reduced erythropoiesis linked to inflammation or illness as a result of the actions of inflammatory cytokines, a condition commonly known as "anemia of chronic disease". The name "anemia of inflammation" is now a more fitting description for this kind of anemia. Overall, inflammatory cytokines cause erythropoiesis to be directly suppressed and have a negative impact on iron availability, which leads to a non-regenerative macrocytic and

normochromic anaemia [32]. These results were in agreement with those proved by a previous report [30].

The inflammatory process that coexists with the majority of respiratory diseases that have been reported in present study may be the cause of leukocytosis and neutrophilia. This was consistent with the findings of a previous report [15]. Thrombocytopenia may result from a number of secondary conditions, such as viral or bacterial infections. This could be linked to a number of thrombocytopenia-related mechanisms, such as immune-mediated destruction, increased splenic clearance, and decreased production, all of which were consistent with a previous study [34]. Instead of being related to antigenic stimulation, the observed lymphocytosis may be caused by epinephrine-associated reactions [20].

Regarding biochemical evaluation, it is well recognized that Proinflammatory cytokines in APR act as messengers between the hepatocytes that synthesis APPs and the illness or disease site. Variations in hepatocyte synthesis of APPs are primarily responsible for variations in their concentration [34]. APPs are thought to be a possible biomarker of the occurrence, severity, and course of inflammation. As a result, some of them have the potential to be helpful instruments for monitoring, diagnosing, and prognosticating various equine illnesses [35, 36].

APPs concentrations can either increase (positive APPs) or decrease (negative APPs) in response to inflammation. The degree to which positive APPs react to inflammation determines which category they fall into. In response to inflammation, the plasma concentrations of major APPs increase more than ten folds, while those of moderate and minor APPs increase two to ten times and less than two-fold, respectively [37]. The APPs (SAA, Hp and CRP) levels were elevated in the current investigation, which may have been caused by the inflammatory process that accompanied the majority of the respiratory diseases that were indicated in our work.

Compared to Hp and CRP, which typically rise and fall more slowly and to a lower extent with inflammation, SAA is regarded as a "major" APP since it increases with inflammation extremely quickly (24–48 hours) and prominent [38]. SAA elevations can rise with inflammation even in the absence of infection and are not exclusive to any particular disease type. Therefore, the physical examination findings and the outcomes of additional diagnostic testing should be considered when evaluating the rise in SAA [39].

SAA has a short half-life; variations in its concentration closely correspond to the initiation of inflammation. SAA concentration is very low, but it

increases quickly with inflammation. As a result, measuring it can help with disease diagnosis, therapy response tracking, and monitoring. It was found to be a more reliable index of a patient's recovery from illness [38]. Because of this characteristic, practical usage of SAA is challenging because even mild inflammatory states can significantly alter SAA levels [39].

Our findings showed a significant rise in SAA concentration that was consistent with the findings of [11, 36, 39]. Compared to Hp and CRP, SAA showed a greater rate and amplitude of rise, suggesting that it is a more sensitive and significant APP in horses.

Haptoglobin was identified as a primary APP in ruminants and has been demonstrated to be a useful indicator of the occurrence and severity of illnesses in cattle, including endocarditis, pneumonia, and mastitis [41]. While in horses, compared to SAA, Hp is a moderate APP that responds to stimuli more slowly [38]. The main function of Hp is binding free haemoglobin to stop iron loss. It starts to rise 12–24 hours after an inflammatory event. Hp has thus been described as a potential marker of chronic inflammatory disease in horses as well as an early indicator of infections in horses [35, 38]. The current study revealed rising Hp levels as reported by previous studies [11,35, 36, 42].

Some Studies in the past have revealed that C-reactive protein (CRP) is a minor APP, which is moderately raised after 3–5 days of an inflammatory event and was increased in horses with pneumonia, enteritis, arthritis [43]. Our study was one of the few studies that measured CRP in equine respiratory diseases at the moment. Our findings revealed elevated CRP levels in the blood of both sick groups; however, upon contrasting these findings with those of other APPs evaluated in the same animals, particularly SAA, our study discovered a significant increase in SAA levels, measuring between 15 and 32 times higher than control values. Although the increase in CRP was only 2 to 10 times, this suggests that SAA is more responsive and sensitive than CRP and is actually thought to be the primary APP in horses. In contrast, CRP responds slowly to an inflammatory process. Consequently, CRP in horses is not seemed to be a reliable indicator of respiratory disorders. This result may be strengthened by previous reports [41,42] who demonstrated that CRP is not a good marker of chronic inflammatory airway disease in horses and has lesser degree of importance in equine than in human, dog and ruminants.

One of the proinflammatory cytokines, or proteins that are chemical messengers produced by inflammatory cells at the site of inflammation, is interleukin-6 (IL-6). It is crucial for the removal of the infection because it promotes phagocytosis and hepatocyte synthesis of APPs. Furthermore, the elevated levels of IL-6 suggest that the animal has

been subjected to multiple stressors and is undergoing immune suppression [34]. Our results revealed increased levels of IL-6 that was agreed with a previous study [50]. This could be due to the inflammatory response and improved the expression of pro-inflammatory factors [43].

According to protein profile analysis, there was a minor rise in globulin and total protein while albumin and the A/G ratio were decreased; these findings were consistent with previous reports [30, 40, 7]. These changes in protein profile during APR may be caused by the increase in synthesis of positive APPs and immunoglobulins. Because the production of positive APPs during the APR uses about 30–40% of the hepatic protein anabolic capacity, this results in a reduction in other proteins such as albumin which is a negative APP that decreased during inflammation. For this reason, hypoproteinemia is typically linked to infection and inflammation [45]. Furthermore, since SAA is one of several APPs that move in the globulin fractions separated by serum protein electrophoresis, a modest negative link between SAA concentration and A/G ratio was found [40]. Additionally, the increased production of immunoglobulins resulted in a drop in the A/G ratio.

Reactive oxygen species (ROS) which are produced *in vivo* have a greater oxidising power than the body's antioxidant capacity, the condition is known as oxidative stress. Relative oxidative stress index (OSI) is known to rise in animals with respiratory disorders as a result of leukocytes such as neutrophils and alveolar macrophages releasing reactive oxygen species (ROS) in response to inflammation [15]. Lipid peroxidation generates a number of relatively stable breakdown end-products such as malondialdehyde (MDA) [46] which can

then be assessed as an indirect indication of oxidative stress. Our findings showed a decline in SOD and CAT activity. Furthermore, there was an increase in MDA, which was consistent with previous reports [17,18]. The reduced levels of SOD activity may be related to the inhibition of the enzyme by free radicals specifically hydrogen peroxide [47]. However, CAT activity dropped, indicating a higher level of oxidative stress. Previous studies [17, 48] have so shown comparable findings. Patients with upper and lower respiratory tract infections showed statistically significant increases in oxidative stress and MDA levels in differing degrees [49]. However, Total Antioxidant Capacity (TAC) decreased in a manner consistent with previous studies [18, 51].

Conclusion

Inflammatory and oxidant markers could be used to monitor the severity and complications of upper respiratory affections in horse, where significant levels were higher in UCL than URT. Therefore, timely intervention is crucial to prevent the progression of upper respiratory affections to lower Respiratory complication, which can have more severe consequences for the horse's health. Regular monitoring and veterinary care are essential for the management of equine Respiratory tract diseases.

Conflicts of Interest

The authors have no conflicts of interest to declare.

Acknowledgements

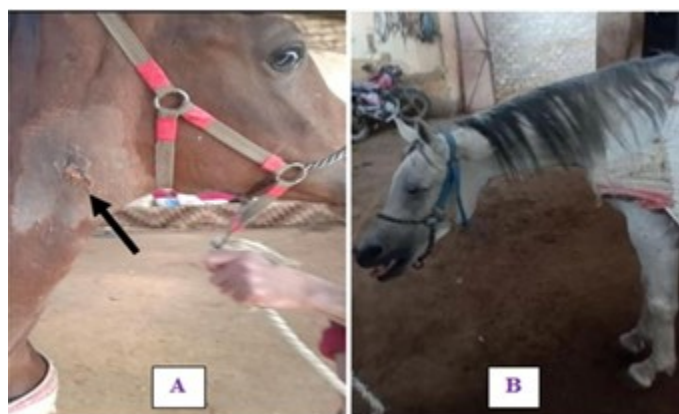
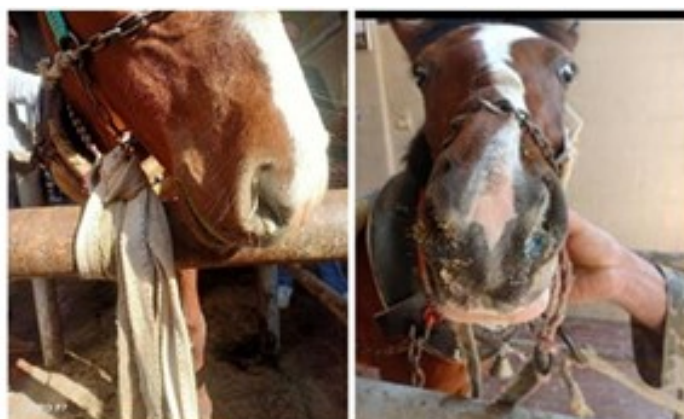
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TABLE 1. The animal experimental design.

Subgroups	Groups	
	Foals (< 1 year old)	Adult horses (2-6 years)
Control	15	15
URT	25	20
UCL	45	30

TABLE 2. Frequency of clinical signs and the ultrasonographic changes of lung in foals and adult horses with upper (URT) and upper complicated with lower (UCL) respiratory affections.

Clinical symptoms	Foal (N = 70)				Adult horse (N = 50)			
	URT N = 25	%	UCL N = 45	%	URT N = 20	%	UCL N = 30	%
Fever	17	68.0	33	73.3	14	70.0	25	83.3
Anorexia	20	80.0	36	80.0	16	80.0	24	80.0
Nasal discharge	16	64.0	27	60.0	15	75	19	63.3
Cough	14	56	35	77.8	9	45.0	22	73.3
Increased respiratory rate	10	40.0	22	48.9	11	55.0	18	60.0
Tachycardia	4	16.0	16	35.6	8	40.0	23	76.7
Congested mucous membrane	6	24.0	18	40.0	8	40.0	16	53.3
Submandibular lymphadenopathy	11	44	14	31.1	6	30.0	11	36.7
Guttural pouch empyema	5	20	0	0	3	15	0	0
Thoracic pain	0	0.0	11	24.4	0	0.0	14	46.7
Ultrasonographic changes	0	0	45	100%	0	0	30	100%

**Fig. 1. (A) Horse with empyema of guttural pouch (URT), (B) Horse showing severe coughing, dullness, depression and dyspnea (UCL).****Fig. 2. Horses with bilateral mucopurulent nasal discharges (URT).**

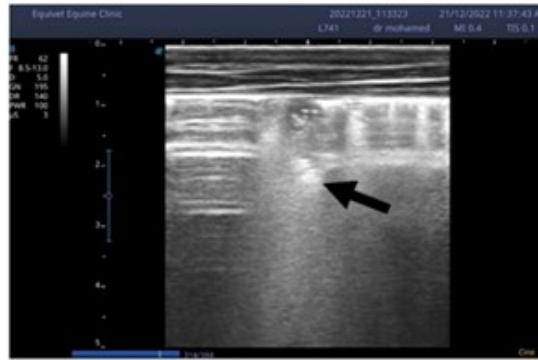


Fig. 3. Ultrasonographic image from the intercostal space of pulmonary area of UCL-affected adult horse showing localized area of lung consolidation (hyperechoic) (arrow).



Fig. 4. Ultrasonographic image from the intercostal space of pulmonary area of UCL-affected adult horse showing localized area of lung consolidation (hyperechoic) (black arrow) and pulmonary exudate (anechoic) (white arrow). Surrounding normal lung tissue also appeared next to the affected areas as reverberation artifacts (small arrows).

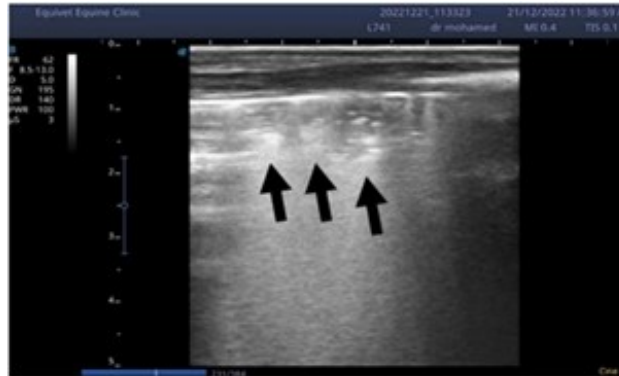


Fig. 5. Ultrasonographic image from the intercostal space of pulmonary area of UCL-affected adult horse showing multiple areas of lung consolidation (hyperechoic) (black arrows).

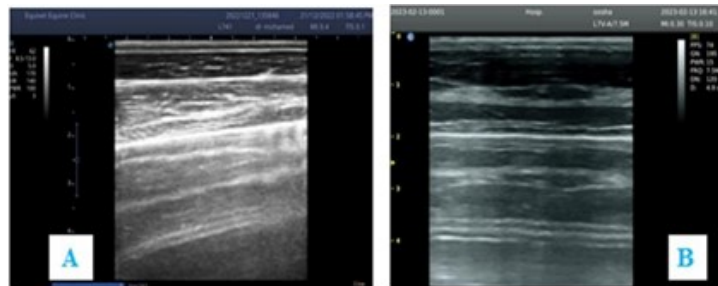


Fig 6. A and B. Ultrasonographic image from the intercostal space of pulmonary area of control healthy adult horse showing the reverberation artefacts without any lung consolidation or fluid.

TABLE 3. Hematological examination of control foals and adult horses with upper (URT) and upper complicated with lower (UCL) respiratory tract affections (Mean ± SE).

Parameters	Subgroups	Groups	
		Foal	Adult
RBCs (x10 ⁶)	Control	9.75±0.13 ^{aA}	10.13±0.52 ^{aA}
	URT	7.30±0.66 ^{bA}	7.90±0.55 ^{bA}
	UCL	6.55±0.89 ^{bA}	6.70±0.37 ^{bA}
Hb (g/dL)	Control	11.18±0.14 ^{aB}	12.50±0.40 ^{aA}
	URT	11.00±0.76 ^{aA}	11.80±0.77 ^{aA}
	UCL	10.00±0.63 ^{bA}	10.50±0.22 ^{bA}
HCT (%)	Control	39.50±0.50 ^{aA}	34.43±1.76 ^{aB}
	URT	30.90±1.55 ^{bB}	33.90±2.06 ^{aA}
	UCL	30.90±1.55 ^{bB}	33.90±2.06 ^{aA}
PLT (x10 ⁶)	Control	2.45±0.25 ^{aA}	2.55±0.06 ^{aA}
	URT	2.00±0.23 ^{bA}	1.01±0.13 ^{bB}
	UCL	1.75±0.23 ^{bA}	1.01±0.13 ^{bB}
WBCs (x10 ³)	Control	6.93±0.29 ^{bA}	8.53±0.32 ^{cA}
	URT	8.90±0.62 ^{bB}	12.82±0.85 ^{bA}
	UCL	13.46±0.54 ^{aB}	21.08±3.05 ^{aA}
Lymph (x10 ³)	Control	2.73±0.11 ^{aA}	3.85±0.12 ^{bA}
	URT	2.76±0.39 ^{aB}	4.36±0.69 ^{bA}
	UCL	3.26±0.31 ^{aB}	6.82±1.88 ^{aA}
Mid (x10 ³)	Control	0.53±0.05 ^{bA}	0.73±0.05 ^{cA}
	URT	0.70±0.04 ^{bB}	1.68±0.47 ^{bA}
	UCL	2.00±0.37 ^{aA}	2.16±0.39 ^{aA}
Neutrophils (x10 ³)	Control	3.63±0.24 ^{cA}	3.90±0.23 ^{cA}
	URT	5.68±0.31 ^{bB}	7.30±0.86 ^{bA}
	UCL	8.20±0.37 ^{aB}	12.10±0.93 ^{aA}

Different small superscript letters indicate significant difference (P<0.05) of means of the same column (subgroups) within each group.
Different Large superscript letters indicate significant difference (P<0.05) of means of the same row between groups.

TABLE 4. Biochemical changes in pro-inflammatory marker and acute phase proteins in young and adult horses suffer from upper (URT) and upper complicated with lower (UCL) respiratory tract affections.

Parameters	Subgroups	Groups	
		Foal	Adult
S.A.A. (mg/L)	Control	2.53±0.09 ^{cA}	2.81±0.09 ^{cA}
	URT	31.00±1.83 ^{bB}	47.89±3.31 ^{bA}
	UCL	42.71±1.92 ^{aB}	65.50±2.79 ^{aA}
Hp (mg/ L)	Control	36.00±0.79 ^{bA}	9.32±0.11 ^{cB}
	URT	46.04±1.66 ^{bA}	15.33±1.38 ^{bB}
	UCL	60.47±1.96 ^{aA}	47.79±4.25 ^{aB}
CRP (g/L)	Control	6.26±0.05 ^{cA}	3.36±0.10 ^{cB}
	URT	9.64±0.89 ^{bB}	14.81±1.49 ^{bA}
	UCL	15.38±1.81 ^{aB}	29.30±1.99 ^{aA}
IL-6 (pg/mL)	Control	1.30±0.17 ^{cB}	2.53±0.09 ^{cA}
	URT	4.00±0.51 ^{bB}	7.83±0.85 ^{bA}
	UCL	8.24±0.56 ^{aB}	11.25±0.68 ^{aA}

Different small superscript letters indicate significant difference (P<0.05) of means of the same column (subgroups) within each group.
Different Large superscript letters indicate significant difference (P<0.05) of means of the same row between groups.

TABLE 5. Biochemical changes in protein profile in young and adult horses suffer from upper (URT) and upper complicated with lower (UCL) respiratory tract affections (mean ± SE).

Parameter	Subgroups	Group	
		Foal	Adult
Total protein (g/L)	Control	7.07±0.11 ^{aA}	6.50±0.03 ^{cB}
	URT	7.20±0.12 ^{aB}	8.89±0.20 ^{aA}
	UCL	7.23±0.21 ^{aB}	8.40±0.18 ^{bA}
Albumin (g/L)	Control	3.84±0.05 ^{aA}	3.21±0.01 ^{aB}
	URT	3.13±0.11 ^{bA}	2.90±0.03 ^{bB}
	UCL	2.40±0.15 ^{cA}	1.73±0.06 ^{cB}
Globulin (g/L)	Control	3.39±0.10 ^{cA}	3.20±0.00 ^{cA}
	URT	4.07±0.05 ^{bB}	5.99±0.20 ^{bA}
	UCL	4.83±0.17 ^{aB}	6.67±0.15 ^{aA}
A/G ratio	Control	1.19±0.04 ^{aA}	1.02±0.00 ^{aB}
	URT	0.77±0.03 ^{bA}	0.49±0.02 ^{bB}
	UCL	0.50±0.04 ^{cA}	0.26±0.01 ^{cB}

Different small superscript letters indicate significant difference ($P < 0.05$) of means of the same column (subgroups) within each group. Different Large superscript letters indicate significant difference ($P < 0.05$) of means of the same row between groups.

TABLE 6. Biochemical changes in oxidant-anti oxidant markers i in young and adult horses suffer from upper (URT) and upper complicated with lower (UCL) respiratory tract affections (mean ± SE).

Parameter	Treatment	Group	
		Foal	Adult
SOD (µg/dL)	Control	19.22±0.07 ^{aA}	15.08±0.11 ^{aB}
	URT	14.66±1.03 ^{bA}	9.02±0.66 ^{bB}
	UCL	11.84±0.90 ^{cA}	4.77±0.72 ^{cB}
MDA (nmol/mL)	Control	13.14±0.13 ^{cA}	4.45±0.23 ^{cB}
	URT	20.55±1.46 ^{bB}	27.23±2.39 ^{bA}
	UCL	32.70±1.93 ^{aB}	45.53±2.86 ^{aA}
TAC (mmol/L)	Control	0.94±0.02 ^{aA}	1.25±0.02 ^{bA}
	URT	0.89±0.03 ^{aA}	1.18±0.34 ^{bA}
	UCL	0.82±0.36 ^{aA}	1.00±0.31 ^{bA}
CAT (mmol/L)	Control	10.45±0.12 ^{aA}	11.03±0.02 ^{aA}
	URT	8.70±0.52 ^{bA}	8.20±0.36 ^{bB}
	UCL	7.35±0.54 ^{cA}	5.43±0.51 ^{cB}

Different small superscript letters indicate significant difference ($P < 0.05$) of means of the same column (subgroups) within each group. Different Large superscript letters indicate significant difference ($P < 0.05$) of means of the same row between groups.

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التغيرات الاكلينيكية والهيماطوبوكيميائية في الأمهار والخيول البالغة ذات الإصابات في القنوات التنفسية

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المستخلص

تعتبر الأمراض التنفسية في الخيول مشكلة شائعة وخطيرة تؤثر على الخيول والامهار على حد سواء. لذلك، هذه الدراسة تهدف إلى تقييم الأمراض التنفسية في الامهار والخيول البالغة ذات الإصابات في مجرى التنفس العلوى والسفلى. تم استخدام إجمالي عدد 150 حصاناً (30 كمجموعة ضابطه و 120 مصاباً) وخضعت للفحص السريري والدموى والكيميائي. تم تصنيف الامهار والخيول إلى مجموعتين: مجموعة مجرى التنفس العلوى (URT) ومجموعة مجرى التنفس العلوى المعقدة بمجرى التنفس السفلى (UCL) وفقاً للفحص السريري الفحص بالموجات فوق الصوتية. تم جمع عينات الدم والمصل من الامهار والخيول المصابة وكذلك المجموعة الضابطة. أظهرت النتائج في مجموعة URT انخفاضاً معنوياً في عدد كرات الدم الحمراء، ونسبة تعبئة الدم، وعدد صفائح الدم، وزلال الدم، ونسبة زلال الدم إلى الجلوبيولين، وفعالية السوبروكسيد ديسموتاز، وفعالية الكاتاليز. أيضاً، كان هناك زيادة معنوية في عدد كرات الدم البيضاء، ونسبة الخلايا المتعادلة، وبروتين سى التفاعلى ، وهابتوجلوبين، وبروتين الاستجابة السريعة، والسيتوكين الرد التنظيمي التفاعلي، والبروتين الكلي، والجلوبيولين، ومادة مؤكسدة مالونديالدهايد، في حين أظهرت مجموعة UCL انخفاضاً كبيراً في عدد كرات الدم الحمراء، ونسبة الهيموغلوبين، ونسبة تعبئة الدم، وعدد صفائح الدم، وزلال الدم، ونسبة زلال الدم إلى الجلوبيولين، وفعالية السوبروكسيد ديسموتاز، وفعالية الكاتاليز. بالإضافة إلى ذلك، كان هناك زيادة معنوية في عدد كرات الدم البيضاء، ونسبة الخلايا المتعادلة، والخلايا الليمفاوية، وبروتين الزلال في المصل، وهابتوجلوبين، والسيتوكين وبروتين سى التفاعلى ، والبروتين الكلي، والجلوبيولين، ومادة مؤكسدة مالونديالدهايد، فضلاً عن كان هناك تغيير كبير في عدد كرات الدم البيضاء، ونسبة الخلايا المتعادلة، والخلايا الليمفاوية، ونسبة تعبئة الدم، وعدد صفائح الدم، و بروتين الزلال في المصل ، وبروتين هابتوجلوبين، والسيتوكين وبروتين سى التفاعلى و البروتين الكلي و زلال الدم والجلوبيولين ونسبة زلال الدم إلى الجلوبيولين و فعالية السوبروكسيد ديسموتاز، وفعالية كاتالاز، بين الامهار والخيول البالغة. وبالتالي، استنتجنا أن التدخل المبكر ضروري لمنع تقدم العدوى من مجرى التنفس العلوى إلى مضاعفات في مجرى التنفس السفلى ، والتي قد تكون لها عواقب أكثر على صحة الخيول.

الكلمات الدالة: المهر- الحصان - مادة مؤكسدة مالونديالدهايد - التنفسى- بروتين الزلال في المصل.